ultimately right heart failure. Previous reports have suggested that Galectin-3 (Gal-3) induced endothelial cell morphogenesis and regulated myofibroblast activation. The aim of this study was to determine the diagnostic utility of circulating Gal-3 as a potential biomarker of disease severity in PAH.

METHODS Gal-3 was measured in plasma from 31 patients with PAH, diagnosed from right heart catheterization, as well as 18 healthy controls by ELISA. Besides, chronic hypoxia induced pulmonary hypertension models were established in Sprague-Dawley rats. Lung tissues were collected for histological analysis including Gal-3 lung qualitative localization by immunohistochemistry. Total mRNA was extracted from pulmonary arteries in rats, then quantitative PCR was performed with total cellular mRNA to measure Gal-3 expression.

RESULTS Plasma level of Gal-3 was significantly decreased in PAH patients compared with healthy controls ($p < 0.001$). Within the subgroups the correlation was given only by idiopathic PAH (IPAH) patients statistically expressed the lower level of Gal-3 ($n = 16$, $p < 0.001$). Gal-3 levels inversely correlated with mean pulmonary arterial pressure (mPAP) ($r = -0.57$, $p = 0.021$) and pulmonary vascular resistance (PVR) ($r = -0.55$, $p = 0.027$), and correlated with cardiac output ($r = 0.530$, $p = 0.035$) in IPAH patients. A Gal-3 cut off value less than 1.765 ng/ml yielded 93% sensitivity and 88% specificity for IPAH patients. Immunohistochemistry method identified Gal-3 was distributed throughout the adventitia of the pulmonary arterioles. The expression of Gal-3 mRNA was significantly down-regulated in the pulmonary arteries from lung tissue samples in pulmonary hypertension rats.

CONCLUSIONS Gal-3 might be involved in the pathogenesis of PAH, and plasma Gal-3 could serve as a promising new biomarker of diagnosis and disease severity in IPAH.

GW26-e3831

Effects of adipose tissue-derived stem cells transfected with adiponectin gene in monocrotaline-induced pulmonary arterial hypertensive rats

Li Luo,[1] Wuhong Zhen,[2] Liangdi Xie[1]
[1]the First Affiliated Hospital of Fujian Medical University, Fuzhou, P.R. China; [2]Fujian Medical University, Fuzhou, P.R. China

OBJECTIVES To observe the effects of adipose tissue-derived stem cells (ADSC) and adiponectin (APN) gene-modified ADSC transplantation in monocrotaline (MCT) induced pulmonary arterial hypertensive (PAH) rats.

METHODS Eighty SD rats were randomly divided into normal control group (NC group), PAH group, ADSC group (ADSC group), empty lentiviral vector infected ADSC group (ADSC-V group) and APN gene modified ADSC (ADSC-APN group). PAH group, ADSC group, ADSC-V group and ADSC-APN group were given 40mg/kg MCT by intraperitoneal injection. Fourteen days after MCT injection, 1.0×10^6 cells/ml suspension, including with ADSC, empty lentiviral vector infected ADSC or APN gene modified ADSC, were injected into ADSC group, ADSC-V group or ADSC-APN group through left jugular vein, respectively. Three weeks after cells transplantation, mean pulmonary arterial pressure (mPAP), right ventricular hypertrophy index (RVHI), vasoconstriction and diastolic function, wall to lumen thickness (WT%) and area (WA%) of pulmonary artery were detected. Serum APN levels were assayed by ELISA. The expression of eNOS in pulmonary artery was detected by immunohistochemical staining. Right heart function was evaluated by echocardiography. Plasmic metabolites were detected by NMR-based metabonomics technology. The expressions of bone morphogenetic protein-2 (BMP2), p-Smad1/5/8, Smad1/5/8/9, p-AMPK and AMPK were detected by western blot after incubation with different combination of Smad inhibitor and AMPK inhibitor on pulmonary arterial smooth muscle cells (PASMCs) of PAH rats. Cells proliferation were detected by CCK-8 kit.

RESULTS Transplantation of ADSC and APN gene-modified ADSC could significantly decrease mPAP, RVHI, WT% and WA%; improve endothelium-dependent and endothelium-independent relaxation of pulmonary artery. Meanwhile, eNOS expression in the intima of pulmonary artery was significantly upregulated. Echocardiography showed that right ventricular structure and function were restored. Compared with ADSC alone transplantation, APN gene-modified ADSC transplantation therapy is better than ADSC transplantation alone. Transplantation of APN gene-modified ADSC could significantly decrease the levels of serum glucose and serum lactate and increase the levels of serum alanine. APN upregulated the BMP2 expression of PASMCs in a dose-dependent manner. BMP inhibitor could reduce the expression of BMP2 and p-Smad1/5/8 induced by APN, and promote cells proliferation. AMPK inhibitor could reduces the expression of p-AMPK and BMP2 induced by APN.

CONCLUSIONS ADSC and APN gene-modified ADSC could increase the expression of eNOS, improve vasodilation, reduce pulmonary artery remodeling, decreased mPAP, and improve right ventricular function. The transplantation of APN gene-modified ADSC was better than ADSC transplantation alone. APN gene-modified ADSC could improve Warburg effect and alanine metabolism in MCT-induced PAH rats. The mechanism that APN inhibits proliferation of PASMCs may be related to the activation of AMPK and BMP/Samd pathway.

GW26-e0216

Systemic arteriovenous shunt porcine model for chronic pulmonary hypertension Functional and Structural Assessment of the Changes in the Pulmonary Circulation

Xuemei Li, Ji Wu
Department of Ultrasound, the First Affiliated Hospital of Guangxi Medical University

OBJECTIVES Altered pulmonary vascular reactivity is a source of morbidity and mortality for children with congenital heart defects (CHD) and increased pulmonary blood flow. The aim of this study was to determine the effects of systemic arteriovenous shunting on the structure and reactivity of the pulmonary circulation in piglets.

METHODS A shunt between the left carotid artery and the jugular vein (CA-JV) was created in 1 month old piglets. Hemodynamic indices were determined periodically by echocardiography to evaluate the pulmonary artery pressure (PAP) and the pulmonary vascular resistance (PVR). Animals were sacrificed at the end of the experiment, 18months after the surgical anastomosis. The lung tissue and plasma were prepared for histopathological and immunohistochemical analysis.

RESULTS The pulmonary arterial systolic pressure (PASP) and mean the pulmonary arterial pressure (MPAP) increased gradually ($P<0.05$) in the process of establishing the chronic pulmonary hypertension model, as did the pulmonary artery acceleration time (PAAT), pulmonary artery deceleration time (PADT) and right ventricle ejection time (RVET). The ratio of PAAT/RVET increased slightly first and then decreased ($P<0.05$). PAAT, PAAT/RVET showed a good correlation with PASP ($r = -0.81, -0.72$). The pulmonary vascular resistance was significantly higher in the shunt-group compared with that of sham-group, while it became higher and higher 10 months after the shunting operations. PAAT and the value of the ratio PAVCR = VPA/(VTIPA × HR) i.e. pulmonary peak velocity (VPA) divided by its velocity time integral (VTIPA) multiplied by the heart rate (HR), showed a good correlation with PVR ($r = -0.68, 0.79$). Morphometric analysis of the pulmonary vascular bed revealed an increase in the number of peripheral muscular arteries, together with an increase in pulmonary arterial medial thickness in the shunt group. The concentration of 3', 5'-cyclic guanosine monophosphate (cGMP) in both the lung tissue and the plasma was enhanced in the shunt-group compared with the controls group.

CONCLUSIONS Systemic arteriovenous shunting leads to both structural and functional alteration of the pulmonary vasculature. PAAT and PAAT/RVET were sensitive indicators to evaluate pulmonary artery hypertension (PAH); PAAT and PAVCR = VPA/(VTIPA × HR) were established as sensitive indicators to evaluate PVR. The elevation of PAP is attributed to the increased PVR. We determined that, with the end-to-side anastomosis of the left carotid artery and the jugular vein in one month old piglets, a good model of pulmonary hypertension with increased pulmonary blood flow can be built.

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In rats with ALI/ARDS, do lung ultrasound results agree with CT imaging results?

Huan Ma, Liheng Guo, Lei Wang, Shuai Mao, Yi Ren, Wenhui Li, Minzhou Zhang
Guangdong Provincial Hospital of Chinese Medicine

OBJECTIVES Lung sonography can diagnose Acute Lung Injury/Acute Respiratory Distress Syndrome (ALI/ARDS) in humans and large animals. To date, no published articles have reported the identification of